CHAPTER 30
Auscultation of the Lungs

KEY TEACHING POINTS

- In patients with chronic dyspnea, diminished breath sounds, when symmetric, increase the probability of chronic obstructive lung disease. Unilateral diminished breath sounds increase probability of underlying pleural effusion or, in patients with cough and fever, pneumonia.
- In patients with cough and fever, egophony and bronchial breath sounds increase probability of pneumonia.
- Crackles can be nonspecific because so many different pulmonary disorders cause them. Nonetheless, in asbestos workers, crackles indicate interstitial fibrosis. In patients with cardiomyopathy, crackles indicate elevated left atrial pressure. Early inspiratory crackles are characteristic of severe chronic airflow obstructive disease.
- Unforced wheezing increases probability of obstructive lung disease, although the amplitude of wheezing correlates poorly with severity of obstruction.

The three categories of auscultatory findings of the lungs are breath sounds, vocal resonance (i.e., the sound of the patient's voice through the stethoscope), and adventitious sounds (i.e., sounds other than breath sounds or vocal resonance). Almost all of the findings discussed in this chapter were originally described in 1819 by Laennec, in his masterpiece *A Treatise on the Disease of the Chest*.[1]

I. BREATH SOUNDS

A. FINDING

1. VESICULAR VERSUS BRONCHIAL BREATH SOUNDS

There are two types of breath sounds: (1) vesicular breath sounds, which are normally heard over the posterior chest, and (2) bronchial breath sounds, which are normally heard over the trachea and right apex. These sounds are distinguished by their timing, intensity, and pitch (Fig. 30.1). Vesicular sounds are mostly inspiratory sounds that have a soft, breathy quality, which Laennec likened to the sound of leaves rustling in a gentle breeze. Bronchial sounds have a prominent expiratory component and much harsher quality, sounding like air blowing forcibly through a tube (hence they are sometimes called tubular breath sounds).

Bronchial breath sounds are abnormal when they occur over the posterior or lateral chest (especially the lower parts). According to traditional teachings, which in turn are based on postmortem examinations, bronchial breath sounds occur in these locations only if solid, collapsed, or consolidated lung is contiguous with the chest wall and extends some distance toward the hilum.[7-9]

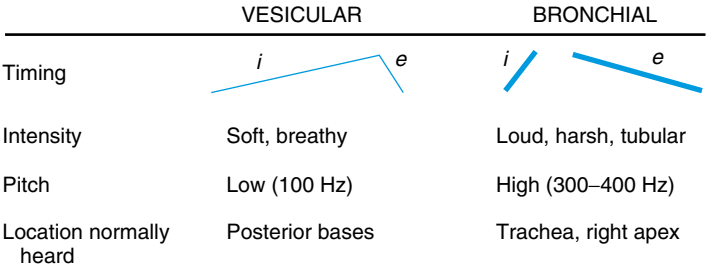


FIG. 30.1 COMPARISON OF VESICULAR AND BRONCHIAL BREATH SOUNDS. In vesicular sounds (*left*), inspiration is longer than expiration, and there is no gap between inspiration and expiration. In bronchial sounds (*right*), expiration is longer than inspiration and there is a conspicuously audible gap between inspiration and expiration. Based upon references 2-6.

The usual causes are pneumonia and pleural effusion (large pleural effusions presumably compress the underlying lung just enough to alter its acoustic properties).[10]

2. BREATH SOUND SCORE

One important feature of vesicular breath sounds is their intensity, which can be graded using a scoring system developed by Pardee.[11] According to this system, the clinician listens sequentially over six locations on the patient's chest: bilaterally over the upper anterior portion of the chest, in the midaxillae, and at the posterior bases. At each site, the clinician grades the *inspiratory* sound as absent (0 points), barely audible (1 point), faint but definitely heard (2 points), normal (3 points), or louder than normal (4 points). The patient's total score may range from 0 (absent breath sounds) to 24 (very loud breath sounds).

B. PATHOGENESIS

1. VESICULAR SOUNDS

A. ORIGIN

The *inspiratory* component of vesicular breath sounds originates in the peripheral portions of the lung near where the stethoscope is placed. It does not represent simple filtration of tracheal sounds by the intervening inflated lung. The *expiratory* component of vesicular sounds probably originates in more proximal, larger airways. Several lines of evidence support these statements.

1. In experiments performed with sheep's and calf's lungs more than a century ago, Bullar kept the airways of both lungs patent but rhythmically inflated only one of the two lungs using negative pressure.[12] He showed that vesicular sounds occurred only if the lung contiguous to the stethoscope filled with air; if it remained airless, it simply transmitted the upper airway bronchial sounds.
2. The intensity of the inspiratory component of breath sounds, corrected for flow rate at the mouth, is approximately proportional to regional ventilation.[13]
3. The inspiratory component of vesicular sounds remains the same as the stethoscope is moved progressively from the upper to lower posterior chest, although the expiratory component becomes softer.[14]

4. Vesicular sounds contain low-frequency components lacking in tracheal sounds, which cannot be reproduced in experiments interposing inflated lung between the trachea and stethoscope.[2-4]

B. INTENSITY

The intensity of vesicular sounds is proportional to the flow rate of air at the mouth, which in turn depends on the patient's effort and ventilatory capacity.[11,15,16] Breath sounds are thus louder if a normal person breathes hard after exercise, and they are faint if obstructive lung disease diminishes flow rates.[17] Breath sounds are also reduced when air or fluid is interposed between the chest wall and lung, as in patients with pneumothorax or pleural effusion.

2. BRONCHIAL SOUNDS

Bronchial breath sounds originate in larger, proximal airways. They are normally heard over the right upper chest posteriorly but not over the left upper chest because the trachea is contiguous with the right lung near the upper thoracic vertebrae but separated from the left lung by most of the mediastinum.[18] The glottis is not necessary to the sound because bronchial sounds may occur in patients after laryngectomy or after intubation.[19] The pathogenesis of bronchial breath sounds in pneumonia and pleural effusion is discussed later in the section entitled Pathogenesis of Vocal Resonance.

C. CLINICAL SIGNIFICANCE

1. BREATH SOUND INTENSITY

A breath sound score of 9 or less greatly increases the probability of chronic airflow obstruction (Likelihood ratio [LR] = 10.2, EBM Box 30.1), and a score of 16 or more greatly decreases the probability (LR = 0.1). The breath sound score is superior to the clinician's "overall impression" of breath sound intensity in diagnosing chronic airflow obstruction (LR = 3.5 for overall impression of "diminished" breath sounds and LR = 0.5 for "normal or increased" breath sounds; see EBM Box 30.1).

Unilaterally diminished breath sounds increase the probability of pleural effusion in hospitalized patients with respiratory complaints (LR = 5.2); in patients with the acute respiratory distress syndrome receiving mechanical ventilation, the absence of breath sounds over a specific region of the chest also increases the probability of underlying pleural fluid (LR = 4.3). In addition, the appearance of reduced breath sounds during methacholine challenge increases the probability of asthma (LR = 4.2), and, in patients with fever and cough, diminished breath sounds modestly increase the probability of pneumonia (LR = 2.2).

The presence of normal breath sound intensity greatly decreases the probability of underlying pleural effusion (LR = 0.1).

2. ASYMMETRIC BREATH SOUNDS AFTER INTUBATION

If the endotracheal tube is placed too low during intubation of a patient, it risks intubating the right mainstem bronchus and leaving the left lung unventilated, a complication that logically would produce asymmetric breath sounds. In studies of patients after intubation, asymmetric breath sounds indeed are pathognomonic for endobronchial intubation (LR = 18.8; see EBM Box 30.1), but the converse is not true: the presence of symmetric breath sounds does *not* significantly decrease the probability of endobronchial intubation (LR = 0.5).

EBM BOX 30.1
*Breath Sounds and Vocal Resonance**

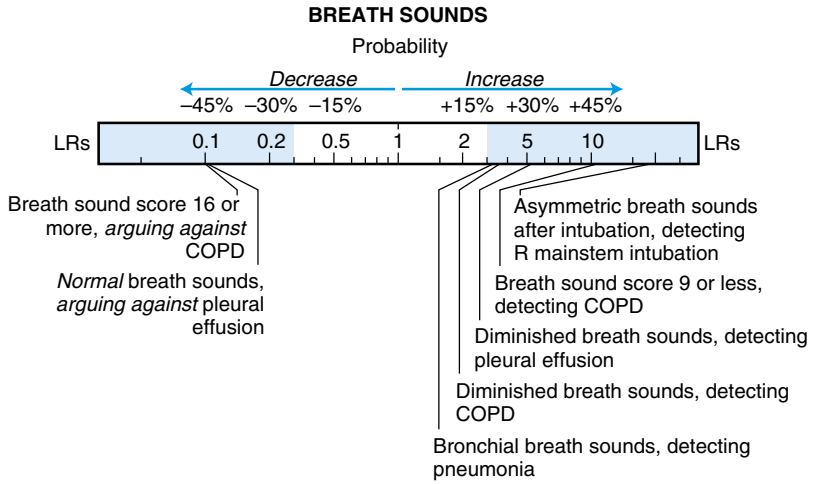
Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	Likelihood Ratio‡ if Finding Is	
			Present	*Absent*
Breath Sound Score				
Detecting chronic airflow obstruction[11,15]				
≤9	23-46	96-97	**10.2**	—
10-12	34-63	—	**3.6**	—
13-15	11-16	—	NS	—
≥16	3-10	33-34	**0.1**	
Diminished Breath Sounds				
Detecting pleural effusion in hospitalized patients[20]	88	83	**5.2**	**0.1**
Detecting chronic airflow obstruction[21-25]	29-82	63-96	**3.5**	0.5
Detecting underlying pleural effusion in mechanically ventilated patient[26]	42	90	**4.3**	0.6
Detecting asthma during methacholine challenge testing[27]	78	81	**4.2**	**0.3**
Detecting pneumonia in patients with cough and fever[28-33]	7-49	73-98	2.2	0.8
Asymmetric Breath Sounds After Intubation				
Detecting right mainstem bronchus intubation[34-36]	28-83	93-99	**18.8**	0.5
Bronchial Breath Sounds				
Detecting pneumonia in patients with cough and fever[28]	14	96	**3.3**	NS
Egophony				
Detecting pneumonia in patients with cough and fever[28,30,37]	4-16	96-99	**4.1**	NS
Diminished Vocal Resonance				
Detecting pleural effusion in hospitalized patients[20]	76	88	**6.5**	**0.3**

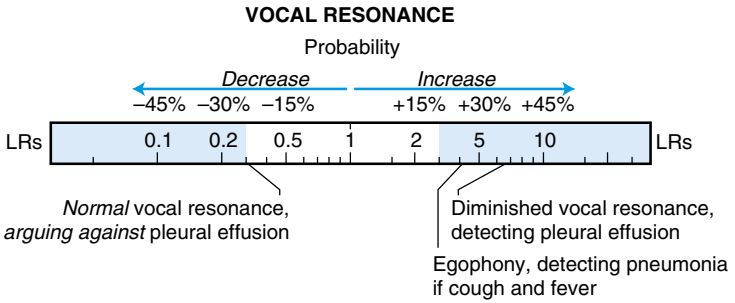
*Diagnostic standard: For *chronic airflow obstruction,* FEV1 <40% predicted (breath sound score) or FEV1:FVC (%) ratio <0.6-0.7 (diminished breath sounds); for *underlying pleural effusion*, chest radiography or (if mechanically ventilated) computed tomography; for *asthma*, FEV1 decreases ≥20% during methacholine challenge; for *pneumonia,* infiltrate on chest radiograph; for *right mainstem intubation*, chest radiograph[34] or direct endoscopic visualization.[35,36]

†Definition of findings: For *breath sound score*, see text; for *diminished vocal resonance intensity*, the transmitted sounds from the patient's voice when reciting numbers, as detected by a stethoscope on the patient's posterior chest, are reduced or absent.[20]

‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.

NS, Not significant.





Confirmation of appropriate tube placement by means other than physical examination is always indicated.

3. BRONCHIAL BREATH SOUNDS

In patients with cough and fever, bronchial breath sounds increase the probability of pneumonia (LR = 3.3), although the sign is infrequent (sensitivity = 14%).

II. VOCAL RESONANCE

A. THE FINDING

Vocal resonance refers to the sound of the patient's voice as detected through a stethoscope placed on the patient's chest. Normally the voice sounds muffled, weak, and indistinct over most of the inferior and posterior chest, and words are unintelligible. Abnormal vocal resonance is classified as either *bronchophony*, *pectoriloquy*, or *egophony*, all terms originally introduced by Laennec.[1] Although these abnormalities have distinct definitions, the pathogenesis for all three is similar, and all may

appear simultaneously in the same patient, frequently accompanied by bronchial breath sounds.

1. BRONCHOPHONY

Bronchophony describes a voice that is much louder than normal, as if the sounds were emitted directly into the stethoscope. The patient's words are not necessarily intelligible.

2. PECTORILOQUY

Pectoriloquy implies that the patient's words are intelligible. Most clinicians test this by having the patient whisper words like "one, two, three"; intelligible whispered speech is called **whispered pectoriloquy**.

3. EGOPHONY

Egophony is a peculiar nasal quality to the sound of the patient's voice, which Laennec likened to the "bleating of a goat."[1] Clinicians usually elicit the finding by having the patient vocalize the long vowel "EE" and then listening for the abnormal transformation of the sound into a loud nasal "AH" (the "AH" sound ranges from the "a" of the word hat to the "a" of the word cart; this finding is sometimes called **E-to-A change**).* Although all vowel sounds are altered by the lung (even healthy lung), what makes egophony distinctive is the intensity of the change and the suddenness with which it appears over a small area on *one* side of the chest.[40] Therefore, before concluding a patient has egophony, the clinician should confirm that a similar change of sound is absent over the identical location of the opposite chest.

B. PATHOGENESIS

Fig. 30.2 depicts the transmission of sound from the larynx to the chest wall in normal persons and in those with pneumonia or pleural effusion. Normal lung behaves like a low-pass filter, which means it easily transmits low-frequency sounds (100 to 200 Hz) but filters out high-frequency sounds (>300 Hz).[6,41-43] Because tactile fremitus (the palpable vibrations on the chest wall from the patient's voice) consists of low-frequency vibrations (100 to 200 Hz), it is a normal finding when symmetric, although tactile fremitus is naturally more prominent in healthy men than healthy women (i.e., men's voices are lower pitched and therefore more likely to generate low-frequency vibrations than women's voices). Tactile fremitus also diminishes as a healthy person sings an ascending scale because the underlying lung resonates less well with higher pitches.

Abnormal vocal resonance (bronchophony, whispered pectoriloquy, and egophony) requires transmission of higher frequencies (>300 Hz) to the chest wall; understanding whispered speech requires the transmission of frequencies of more than 400 Hz (i.e., whispered pectoriloquy). The sound "AH" contains more high-frequency energy than the sound "EE," and if the underlying lung preferentially amplifies the high-frequency energy of a vocalized "EE," it may render it into a nasal "AH" (i.e., egophony).[6,42] Because the normal lung does not transmit high-frequency (>300 Hz) sounds well, especially to the lower posterior and lateral chest, egophony and bronchial breath sounds at these locations always

*The E-to-A change was simultaneously discovered in 1922 by Shibley[38] and Fröschel.[39] Shibley discovered it while testing for pectoriloquy in Chinese patients. He asked the patients to say "one, two, three" in the local dialect (ee, er, san), and he noted that the long "EE" of "one" acquired a loud nasal "AH" quality over areas of pneumonia or effusion.[38]

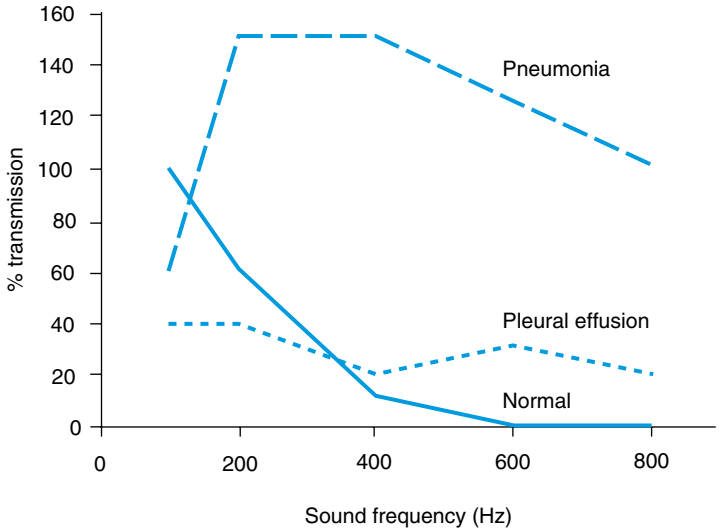


FIG. 30.2 TRANSMISSION OF SOUND TO THE CHEST WALL. In this experiment a speaker emitting pure musical tones of different frequencies was placed in the mouth of patients with normal lungs (*solid line*), pneumonia (*long dashes*), or pleural effusion (*short dashes*). Microphones on the chest wall recorded the transmission of each frequency (for purposes of comparison, 100% transmission is the transmission of 100 Hz in normal persons). Based upon reference 41.

indicate the presence of *abnormal* lung between the patient's vocal cords and clinician's stethoscope.

According to Fig. 30.2, consolidated lung transmits both high and low frequencies well, thus explaining why patients with pneumonia may simultaneously exhibit both increased tactile fremitus and abnormal vocal resonance (i.e., egophony). In contrast, moderate or large pleural effusion may *decrease* transmission of frequencies below 200 to 300 Hz but *augment* those greater than 400 Hz, compared with normal lung (see Fig. 30.2).[6,10,41-43] This explains why some patients with pleural effusion exhibit both *decreased* tactile fremitus yet *abnormal* vocal resonance (i.e., egophony).

Nonetheless, the finding of egophony (abnormal vocal resonance) in patients with pleural effusion is an inconstant finding, and many patients instead demonstrate *reduced* or *absent* vocal resonance over the affected side (i.e., the patient's spoken voice is inaudible or markedly diminished and the nasal "AH" is absent). Laennec himself taught that egophony is not always present pleural effusion but first appears when effusions are moderate in size, then *disappears* as effusions continue to grow larger, and finally *reappears* as effusions began to resolve.[1] The conventional explanation for these findings is that atelectatic lung, resting on top of an effusion, remains close enough to the chest wall to preferentially conduct enough high-frequency sound to produce abnormal vocal resonance (loudest near the angle of the scapula); as effusions continue to grow larger, the distance between compressed lung and chest wall increases and egophony thus disappears.

Nonetheless, this explanation has never been verified, and it remains a mystery why some patients with effusion have prominent egophony over large areas of the

posterior chest wall yet others have diminished vocal resonance. The only study of this finding shows that pleural effusions producing abnormal vocal resonance (e.g., egophony) have higher positive intrapleural pressures than effusions without the finding.[10] From an acoustic standpoint, the variables responsible for abnormal vocal resonance might include not only the size of effusion and condition of the underlying compressed lung but also the amount of air moving in and out of the underlying lung, the viscosity of the pleural fluid, and the condition of the underlying inflamed pleural surface and chest wall.

C. CLINICAL SIGNIFICANCE

Abnormal vocal resonance has the same significance (and pathogenesis) as bronchial breath sounds. In patients with cough and fever, the finding of egophony increases the probability of pneumonia (LR = 4.1; EBM Box 30.2), and in hospitalized patients with a variety of respiratory complaints, the finding of diminished vocal resonance (i.e., diminished intensity of patient's voice when reciting numbers) increases the probability of an underlying pleural effusion (LR = 6.5).

According to traditional teachings an obstructed bronchus should diminish vocal resonance, although this teaching is probably incorrect, based on the observation that some patients with egophony and pneumonia have obstructed bronchi from tumors,[42] and on experiments showing that sound conducts down the substance of the porous lung itself to the chest wall, not down the airway ducts.[†63]

III. ADVENTITIOUS SOUNDS

A. INTRODUCTION

Adventitious sounds are all sounds heard during auscultation other than breath sounds or vocal resonance. The common adventitious sounds are crackles, rubs, wheezes, rhonchi, and stridor.

Adventitious sounds have the most ambiguous and confusing nomenclature in all of physical diagnosis, and studies show clinicians use up to 16 different terms in scientific publications to describe similar sounds.[64] This confusion stems from the earliest days of auscultation and the writings of Laennec, who, in the first edition of his treatise, identified five adventitious sounds but called them all *rales*, distinguishing them further only by adding adjectives (e.g., "moist crepitus rale" for a crackling sound or "dry sibilus rale" for a whistling sound).[1,65] In later editions Laennec substituted *rhonchus* for *rale* because he became worried that patients hearing *rale* would mistake it for the death rattle (*rale* means rattle). In 1831 a British editor introduced the Anglo-Saxon term *wheeze*, again to refer to all lung sounds.[65] Finally, Robertson in 1957 proposed using *crackling sounds* for discontinuous sounds and *wheeze* for continuous, musical sounds, and suggested eliminating *rale* and *rhonchus* altogether.[66]

According to the American Thoracic Society the recommended terms for lung sounds, based on their acoustic characteristics,[67] are **crackle** for discontinuous sounds and **wheeze** or **rhonchus** for continuous sounds (Table 30.1).

[†]The acoustic characteristics of the transmitted sound are the same whether the patient breathes air or a mixture of oxygen and helium. If sound were conducted down the airways, its characteristics would change with different gas mixtures.[63]

EBM BOX 30.2
*Crackles and Wheezes**

Finding (Reference)	*Sensitivity (%)*	*Specificity (%)*	Likelihood Ratio† if Finding Is	
			Present	*Absent*
Crackles				
Detecting pulmonary fibrosis in asbestos workers[44]	81	86	**5.9**	**0.2**
Detecting elevated left atrial pressure in patients with cardiomyopathy[45-48]	15-64	82-94	2.1	NS
Detecting myocardial infarction in patients with chest pain[49,50]	20-38	82-91	2.1	NS
Detecting pneumonia in patients with cough and fever[28-33,37,51,52]	19-67	36-96	2.3	0.8
Early Inspiratory Crackles				
Detecting chronic airflow obstruction in patients with crackles[53,54]	25-77	97-98	**14.6**	NS
Detecting severe disease in patients with chronic airflow obstruction[54]	90	96	**20.8**	**0.1**
Unforced Wheezing				
Detecting chronic airflow obstruction[21,23,25,55-58]	13-56	86-99	2.6	0.8
Detecting pneumonia in patients with cough and fever[28-32,51,52]	10-36	50-85	0.8	NS
Detecting pulmonary embolism[59-61]	3-31	68-91	0.4	NS
Wheezing During Methacholine Challenge Testing				
Detecting asthma[27]	44	93	**6.0**	0.6
Pleural Rub				
Detecting pulmonary embolism[61,62]	1-14	91-99	NS	NS
Detecting pleural effusion[20]	5	99	NS	NS

*Diagnostic standard: For *pulmonary fibrosis,* fibrosis on high resolution computed tomography; for *elevated left atrial pressure*, pulmonary capillary wedge pressure >20 mm Hg[46,47] or >22 mm Hg;[45,48] for *myocardial infarction,* development of new electrocardiographic Q waves, elevations of cardiac biomarkers (CK-MB or troponin), or both; for *pneumonia,* infiltrate on chest radiograph; for *chronic airflow obstruction,* FEV1:FVC <0.6,[21] <0.7,[23,25,55] <0.75,[54] or less than lower 95% confidence interval for age, gender, and height;[53,56-58] for *severe obstruction,* FEV1:FVC <0.44;[54] for *asthma,* FEV1 decrease ≥20% during methacholine challenge;[27] for *pulmonary embolism*, see Chapter 34; and for *pleural effusion*, chest radiograph.

†Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.

NS, Not significant.

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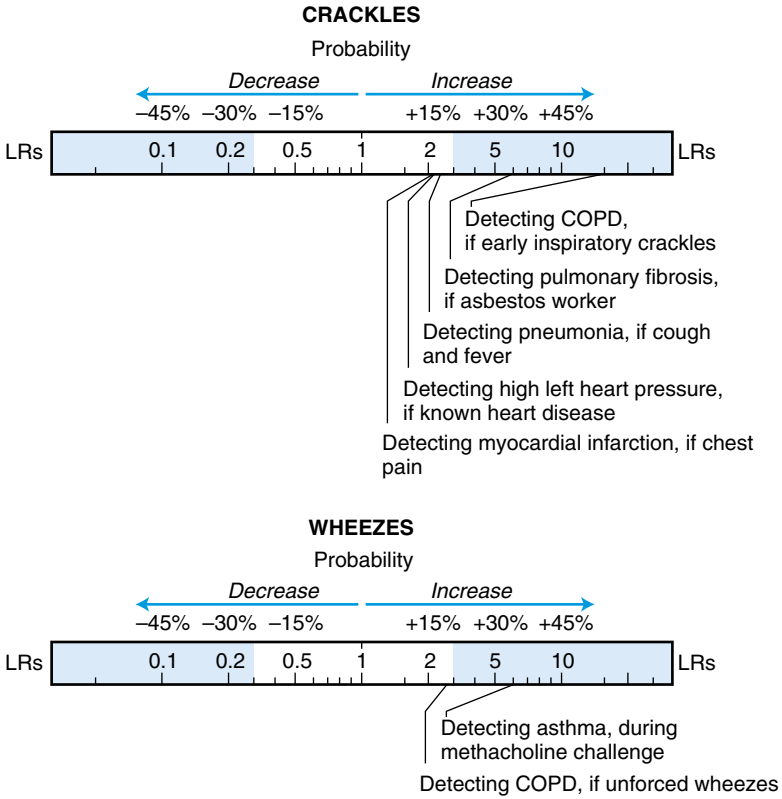

B. THE FINDING

1. CRACKLES

Crackles are discontinuous sounds, resembling the sound produced by rubbing strands of hair together in front of the ear or by pulling apart strips of Velcro. There are **coarse crackles**, which are loud, low pitched, and fewer in number per breath, and **fine crackles**, which are soft, higher pitched, and greater in number per breath. Crackles that appear early during inspiration and do not continue beyond mid-inspiration are called **early inspiratory** crackles; those that continue into the second half of inspiration are called **late inspiratory** crackles.[54] Many American clinicians still use the word *rale* as a synonym for crackle, although British clinicians more often use crackle.[70,71]

The finding **posturally induced crackles**, which may have significance after myocardial infarction (see the section on Clinical Significance, later), describes crackles that appear in the supine position but disappear in the sitting position. To elicit the finding, the clinician listens to the lower chest wall near the posterior axillary line with the patient in three sequential positions: sitting, supine, and supine with legs elevated 30 degrees.[72] The clinician listens only after the patient has been in

TABLE 30.1 Terminology for Lung Sounds

Recommended ATS Term	Acoustic Characteristics	Terms in Some Textbooks	British Usage
Coarse crackle	Discontinuous sound: loud, low in pitch	Coarse rale	Crackle
Fine crackle	Discontinuous sound: soft, higher pitch, shorter duration	Fine rale	Crackle
Wheeze	Continuous sound: high-pitched, dominant frequency ≥400 Hz	Sibilant rhonchus	High-pitched wheeze
Rhonchus	Continuous sound: low-pitched, dominant frequency ≤200 Hz	Sonorous rhonchus	Low-pitched wheeze

Based upon references 67-69.
ATS, American Thoracic Society.

each position for 3 minutes. If crackles are absent when upright but appear either when supine or with legs elevated, the test is positive (i.e., the patient has posturally induced crackles).

2. WHEEZES AND RHONCHI

According to the American Thoracic Society a wheeze is a high-pitched, continuous musical sound and a rhonchus is a low-pitched one (see Table 30.1). This distinction may be superfluous because both sounds have the same pathophysiology and there is no proven clinical importance to separating them. The term *rhonchus* is probably best avoided, not only for these reasons but because many use the term to refer to the coarse discontinuous sounds heard in patients with excess airway secretions.[70]

3. STRIDOR

Stridor is a loud, musical sound of definite and constant pitch (usually about 400 Hz) that indicates upper airway obstruction.[43,69] It is identical acoustically to wheezing in every way except for two characteristics: (1) stridor is confined to inspiration, whereas wheezing is either confined entirely to expiration (30% to 60% of patients) or occurs during both expiration and inspiration (40% to 70% of patients);[73,74] and (2) stridor is always louder over the neck, whereas wheezing is always louder over the chest.[74]

In some patients with upper airway obstruction, stridor does not appear until the patient breathes rapidly through an open mouth.[75]

4. PLEURAL RUB

Pleural rubs are loud grating or rubbing sounds associated with breathing that occur in patients with pleural disease. Sometimes, a pleural rub has a crackling character (**pleural crackling rub**) and acoustically resembles the crackles heard in patients with parenchymal disease.[76,77] The timing of the crackling sound best distinguishes the pleural crackling rub from parenchymal crackles: the pleural crackling rub is predominately *expiratory* (i.e., 65% of crackling sound occurs during expiration) but parenchymal crackles are predominately *inspiratory* (i.e., only 10% of crackling sound occurs during expiration).[78]

5. INSPIRATORY SQUAWK

The **squawk** is a short, late inspiratory musical sound associated with parenchymal crackles in patients with interstitial lung disease,[79] although the sound has also been described in pneumonia.[80] It is best heard over the upper anterior chest when the patient is semirecumbent and breathing deeply. Because the sound is sometimes found in patients with bird fancier's lung (a cause of hypersensitivity pneumonitis), the synonym **chirping rale** has been proposed.[81]

In patients with hypersensitivity pneumonitis the squawk tends to be shorter, higher pitched, and later in inspiration than the squawk of patients with diffuse pulmonary fibrosis.[79]

C. PATHOGENESIS

1. CRACKLES[43,54,76,82-84]

Crackles were initially attributed by Laennec and early auscultators to air bubbling through airway secretions. Although some crackles result from secretions, these promptly clear after the patient coughs. All remaining crackling sounds are felt to represent the sounds of distal airways, collapsed from the previous exhalation, as they abruptly open during inspiration. Several lines of evidence support this conclusion: (1) crackles are predominantly heard during inspiration, whereas air bubbling though secretions would cause both inspiratory and expiratory sounds; (2) the number of crackles has no relationship to the amount of sputum the patient produces (the disease with the most crackles, interstitial fibrosis, produces scant sputum or no sputum at all);[85] (3) crackles have a stereotypic pattern with each respiratory cycle (i.e., in a single patient at a single location on the chest, they are always early, late, or pan-inspiratory, and individual crackles occur at the same esophageal (transpulmonary) pressure in consecutive respiratory cycles;[86] and (4) crackles are loudest in the lower portions of the chest, even when the lung disease is distributed diffusely.

Course crackles are felt to originate in larger, more proximal airways than fine crackles, based on the observations that distinct patterns of coarse crackles (identified by their fingerprint of identical timing and number) radiate to a larger area of the chest wall than do distinct patterns of fine crackles.[87,88]

2. WHEEZES

Wheezes are caused by vibrations of the opposing walls of narrowed airways.[76,82,89] They are not due to resonance of air in the airways (i.e., like the sound of a flute or pipe organ) for the following reasons: (1) if they were due to resonance of air in a hollow pipe, the length of pipe for some low-pitched wheezes would be several feet, far exceeding the length of human airways; (2) the pitch of a wheeze may change between inspiration and expiration; and (3) the pitch of the wheeze remains the same when inspired air is replaced with a gas mixture of oxygen and helium. (If due to resonance of air, the pitch should change.)

D. CLINICAL SIGNIFICANCE

1. CRACKLES

The crackles discussed below refer only to crackling sounds that persist after the patient coughs.

A. NORMAL PERSONS

Crackles are rare in healthy persons during normal tidal breathing.[90,91] However, fine crackling sounds may appear in up to 60% of healthy persons, especially over the anterior chest, if the person first exhales as much as possible and breathes in from residual volume instead of functional residual capacity.[90,91]

B. CRACKLES AND DISEASE

(1). PRESENCE OF CRACKLES. EBM Box 30.2 indicates that the finding of crackles increases the probability of pulmonary fibrosis in asbestos workers (LR = 5.9), of pneumonia in patients with cough and fever (LR = 2.3), of elevated left atrial pressure in patients with known heart disease (LR = 2.1), and of myocardial infarction in patients with chest pain (LR = 2.1). In the evaluation of patients for either pulmonary embolism or pleural effusion, the finding of crackles is unhelpful (LRs not significant; see Chapters 34 and 35).

Some interstitial lung diseases produce more crackles than others. For example, crackles are found in 100% of patients in idiopathic pulmonary fibrosis but only 5% to 20% of patients with fibrosis from sarcoidosis.[85,92] This suggests that the *absence* of crackles *decreases* the probability of idiopathic pulmonary fibrosis. The only finding from computed tomography that seems to predict crackles in interstitial fibrosis is the degree of subpleural fibrosis.[92]

Although the finding of posturally induced crackles after myocardial infarction has been associated with higher pulmonary capillary wedge pressures and worse survival,[72] it is clear that any crackles in patients with acute coronary syndromes portends a worse prognosis. In one study of patients with acute sustained ischemic chest pain, crackles predicted 30-day mortality with a sensitivity of 36%, specificity of 92%, and a positive LR of 4.5.[93] The extent of crackles in patients with newly diagnosed congestive heart failure also predicts future cardiovascular mortality.[94]

(2). CHARACTERISTICS OF CRACKLES.[53,78,95-97] Table 30.2 describes the characteristic number, timing, and type of crackles in common crackling disorders, such as pulmonary fibrosis, congestive heart failure, pneumonia, and chronic obstructive lung disease. The crackles of interstitial fibrosis are characteristically fine, have

TABLE 30.2 Characteristics of Crackles in Various Disorders*

Diagnosis	Number of Crackles per Inspiration	Timing of Crackle	Type of Crackle
Pulmonary fibrosis	6-14	Late inspiratory (0.5 → 0.9)	Fine
Congestive heart failure	4-9	Late or pan-inspiratory (0.4 → 0.8)	Coarse or fine
Pneumonia	3-7	Pan-inspiratory (0.3 → 0.7)	Coarse
Chronic airflow obstruction	1-4	Early inspiratory (0.3 → 0.5)	Coarse or fine

**Number of crackles* is mean number of crackles ± 1 standard deviation, after the patient first coughs to clear airway secretions. The descriptors *early inspiratory, late inspiratory, pan-inspiratory, coarse,* and *fine* are observations made by clinicians listening with the stethoscope; the numbers under *timing* refer to when crackles begin and end during a full inspiration (e.g., 0.5 → 0.9 means that crackles first appear at mid-inspiration [0.5] and end when the patient has reached 90% of full inspiration [0.9].) Based on references 53, 78, and 95.

a large number of individual crackling sounds each inspiration, and persist to the end of inspiration (i.e., they are late inspiratory crackles). Crackles of chronic airflow obstruction are coarse or fine, have the smallest number of crackling sounds, and are confined to the first half of inspiration (early inspiratory crackles). The crackles of heart failure and pneumonia lie between these extremes; with treatment, the crackles of pneumonia become finer and move toward the end of inspiration.[96,97]

EBM Box 30.2 indicates the finding of early inspiratory crackles greatly increases the probability of chronic obstructive lung disease (LR = 14.6). Most patients with these crackles have severe obstruction (LR = 20.8).

2. WHEEZES

A. PRESENCE OF WHEEZES

EBM Box 30.2 indicates that the finding of unforced wheezing increases the probability of chronic obstructive lung disease a small amount (LR = 2.6) and decreases the probability of pulmonary embolism (LR = 0.4). If wheezing appears during methacholine challenge testing, asthma is likely (LR = 6.0). The absence of wheezing in any of these settings is unhelpful.

In contrast, the finding of *forced* wheezing lacks diagnostic value because it can be produced by most healthy persons if they exhale forcibly enough.[55,98]

B. CHARACTERISTICS OF WHEEZING

The characteristics of wheezes are their length, pitch, and amplitude. Of these, only length and pitch vary with severity of obstruction. The longer the wheeze, the more severe the obstruction ($r = -0.89$ between the proportion of the respiratory cycle occupied by wheezing and the patient's FEV1,‡ $p < 0.001$).[73,99,100] Higher-pitched wheezes indicate worse obstruction than lower-pitched ones, and effective bronchodilator therapy reduces the pitch of the patient's wheeze.[73,99]

However, the amplitude of the wheeze does not reflect the severity of obstruction, principally because many patients with severe obstruction have faint or no wheezes.[55,73,99,100] This finding supports the old adage that, in a patient with asthma, the quiet chest is not necessarily a favorable sign but may instead indicate a tiring patient who is unable to push air across the obstructed airways.

The **slide whistle sound**, a unique wheezing sound whose pitch rises during inspiration and falls during expiration, has been described in a patient with a spherical tumor arising from the carina that nearly completely obstructed the trachea.[101]

3. STRIDOR

In patients with tracheal stenosis after tracheostomy, stridor is a late finding, usually appearing after symptoms like dyspnea, irritative cough, or difficulty clearing the throat.[75] Stridor indicates that the airway diameter is less than 5 mm.[75]

4. PLEURAL RUB

EBM Box 30.2 indicates that the presence or absence of a pleural rub does not change the probability of pulmonary embolism or pleural effusion.

The references for this chapter can be found on www.expertconsult.com.

‡See Chapter 28 for definition of FEV1.

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